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The Effect of Feedback Sensitivity on Learned Heart Rate Acceleration and Deceleration.

John Edward Monguillot

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**THE EFFECT OF FEEDBACK SENSITIVITY ON LEARNED HEART RATE
ACCELERATION AND DECELERATION**

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THE EFFECT OF FEEDBACK SENSITIVITY
ON LEARNED HEART RATE ACCELERATION AND DECELERATION

A Dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy

in

The Department of Psychology

by
John Edward Monguillot
B.S., Louisiana State University, 1975
M.A., Louisiana State University, 1979
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ABSTRACT

The effects of sensitivity of heart rate feedback on bidirectional heart rate control were examined by comparing two groups of 10 subjects who received feedback signals of either high or low sensitivity. Subjects in the high sensitivity condition received a feedback signal in which 1 cm of feedback meter needle movement was equivalent to an 8.57 beat-per-minute (BPM) change in momentary heart rate. The low sensitivity condition received a feedback signal which was one-half as sensitive, i.e. 1cm = 17.14 BPM change. All subjects received 8 sessions of training and each session included three within-session phases. The three within-session phases consisted of two (pre- and post-Feedback) Instructional Control phases during which subjects were instructed to accelerate or decelerate heart rate without the aid of feedback, and a Feedback phase during which bidirectional heart rate control was assisted by analogue heart rate feedback. Heart rate, frontal EMG, and respiration rate were recorded during all phases.

The results showed that the low sensitivity condition produced larger mean heart rate accelerations than did the high sensitivity condition across all sessions and in the final session. No significant differences between the sensitivity conditions were found for heart rate

decelerations or for the within-session phases. No differences between the sensitivity conditions were seen in frontal EMG or respiration rate.

These findings were discussed in terms of Brener and Lang's models of learned heart rate control. Both models received some support from the present findings but it was concluded that both models are in need of revision. Possible effects of situational and biological constraints and motivational influences on this study's findings were examined. Finally, these findings were discussed in terms of implications for heart rate biofeedback research and applications.

INTRODUCTION

The study of voluntary heart rate control in humans has gone through three phases which are distinct conceptually but which overlap chronologically. The first phase addressed the question of whether humans could learn to control heart rate voluntarily. The studies of this first phase were designed to test whether instrumental conditioning procedures could be used to produce heart rate increases and decreases (Brener, 1966; Brener & Hothersall, 1966; Engel & Hansen, 1966; Kimmel, 1967). The most important review of this literature (Katkin & Murray, 1968) highlighted the concern for controlling somatic responses, such as respiration, which subjects could use to modify heart rate.

The concern for controlling such somatic responses was based upon a conceptualization of learned autonomic control which viewed changes in autonomic functions as possibly being caused by changes produced in related voluntary responses. For example, if a subject learned to increase heart rate during heart rate biofeedback training and also showed increases in muscle tension, perhaps the subject produced changes in heart rate by increasing muscle tension. Where this conceptualization views one peripheral response as causing changes in another peripheral response, an

alternative conceptualization has been put forth more recently (Brener, 1974a; Obrist, 1976; Schwartz, 1974). This viewpoint considers all peripheral responses to be integrated and controlled at higher neural centers, presumably the brain. The common observation that any behavioral response is accompanied by other simultaneous behavioral changes is taken as support for the second conceptualization (Williamson & Blanchard, 1979b). As this conceptualization proposes that attempts to learn to change heart rate will be accompanied by changes in other responses, e.g. muscle tension and/or respiration, the question of whether one response produces changes in another response is replaced by a different question, i.e. can a specific cardiovascular response be modified voluntarily? The question of the specificity of the response being voluntarily modified has been expressed in terms of the relationship between the targeted response and simultaneous changes in non-targeted responses (Brener, 1974a). Specificity of response modification is defined as a reduction or elimination of the simultaneous non-targeted changes across training while maintaining or improving changes in the targeted response. As a result of the more recent question of specificity of response modification, there has been a trend in the literature to measure multiple physiological responses in order to assess changes across training in these non-targeted responses. A definitive conclusion

concerning the specificity of changes in learned cardiovascular control awaits further study (Williamson & Blanchard, 1979b).

Although the issue of the specificity of changes in learned cardiovascular control remains unanswered, by the early 1970's it became generally accepted that humans could learn to control their heart rates voluntarily (Blanchard & Young, 1973). The second phase of studying voluntary heart rate control focused on determining what parameters influenced the magnitude of heart rate change produced. A number of procedural variables have been found which affect learning voluntary heart rate control. In their review article, Williamson and Blanchard (1979b) noted five such procedural variables. First, informing subjects that heart rate is the response to be controlled facilitates learning to increase and decrease heart rate. Some early work by Engel and his associates failed to inform subjects that heart rate was the response to control (Engel & Hansen, 1966) and a retrospective analysis of this data showed that knowing heart rate was the response of interest was associated with failure to learn to decrease heart rate. A number of studies have been reported which have used knowledge of the response to be controlled as an independent variable (Bergman & Johnson, 1972; Blanchard, Scott, Young, & Edmundson, 1974; McCanne & Sandman, 1975, 1976). Contrary to the conclusions of Engel and Hansen (1966), these studies have shown that knowledge of the response to be controlled

facilitates learning to increase and decrease heart rate. Second, the type of feedback influences the degree of learned heart rate control. Two general types of feedback have been used, binary feedback and analogue feedback. Binary feedback provides only information about whether or not a criterion has been met. Analogue feedback provides information about the degree and direction of heart rate change produced. Analogue heart rate feedback has been found to be superior to binary heart rate feedback for learning to produce heart rate increases. For producing heart rate decreases, analogue heart rate feedback has not been shown to be consistently superior to binary heart rate feedback. Third, immediate analogue heart rate feedback has been found to be superior to delayed (5 seconds or more) analogue heart rate feedback for learning both heart rate speeding and slowing. Fourth, feedback training which extends beyond one or two sessions appears to increase the likelihood of obtaining large magnitude heart rate increases. Extended training has an inconsistent relationship with learning to produce heart rate decreases. Fifth, feedback which is provided on a beat-by-beat basis is superior to feedback provided less frequently when learning to increase heart rate. Beat-by-beat feedback and less frequent feedback have been found to be equivalent when learning to decrease heart rate. Two other less powerful procedural variables, monetary incentives and temporal spacing of training, were also reviewed. The use of

monetary incentives may improve heart rate control, but this finding has been inconsistently supported. The distribution of training sessions over time has little effect on learning to control heart rate.

The third phase of the study of voluntary heart rate control in humans has involved the development of theoretical models to explain learned heart rate control. Following Engel's (1972) suggestion that learned heart rate control might best be considered as similar to learning a motor skill, theoretical models developed by Schwartz, Brener, and Lang have incorporated motor skills concepts. The motor skills literature deals with the development of skilled performance in motoric activities such as lever positioning, tracking, and sports. Within the motor skills literature, an emphasis is placed upon the role of feedback in assisting the development of skilled motor performance.

Schwartz (1974, 1975, 1977) makes little use of motor skills principles in his model although he notes that he prefers that viewpoint (1974, p. 440). He has emphasized the patterning of physiological responses in his model. This model predicts that the response patterns learned during biofeedback training will result from an interaction between the specific feedback contingency used and biological constraints in the physiological system(s) being modified. As an example of the effect of feedback contingencies, if systolic blood pressure and heart rate were related over time such that increases in one were

always associated with increases in the other, then feedback presented for one response, e.g. heart rate increases, would always be presented for the other response. As a result, if positive feedback was presented for heart rate increases, this feedback would also be contingent on systolic blood pressure increases. It would be expected that increases of both responses would be learned in this example, even though only feedback for one was presented. Patterns of more than one response might also be used as criteria for presenting feedback, e.g. green light is lighted whenever heart rate is decreased at the same time that diastolic blood pressure is increased. Schwartz (1974) notes that learning such patterns of responses operates within the context of biological constraints. That is, there may be physiological constraints which prevent or retard learning to produce a particular pattern of responses. Even though two responses might not be related in time so that feedback contingent on one is also contingent on the other, there may be physiological constraints which mitigate against learning a particular pattern of responses. Using different patterns of feedback and measuring changes in the responses being monitored, Schwartz and his associates (Schwartz, 1972; Schwartz, Shapiro & Tursky, 1972; Hassett & Schwartz, 1975) have attempted to document some of these physiological constraints. Schwartz's model focuses on patterning of responses and physiological constraints, but does not deal

with parameters of response feedback, which is one of the main values of motor skills theory.

Lang's theoretical formulations (1974, 1975) have focused on differences in learning to increase heart rate as opposed to learning to decrease heart rate. Several studies performed by Lang and his associates found that certain feedback parameters affected learning to speed heart rate but these parameters showed no reliable effect on learning to slow heart rate. The parameters examined were frequency of feedback (Gatchel, 1974), monetary incentives (Lang & Twentyman, 1976), analogue vs. binary feedback (Lang & Twentyman, 1974), and feedback timing (Twentyman & Lang, 1980). Based on these results, Lang proposed that learning to increase heart rate should be conceptualized as learning a motor skill and that predictions based on the literature of motor skills training would apply to learning heart rate speeding. Lang also proposed that learning to decrease heart rate represents autonomic learning. He differentiates these types of learning on the basis of the physiological mechanisms involved in each. Lang suggested that learning to increase heart rate is dependent upon central linkages which couple somatic and cardiovascular systems while learning to decrease heart rate is relatively independent of somatic activity.

Two predictions may be derived from Lang's position (Williamson & Blanchard, 1979c). First, parameters which have been shown to systematically affect motor learning

should also affect learning to speed heart rate. Second, these same parameters should not affect learning to slow heart rate. Along with the previously mentioned studies which support these predictions, Blanchard, Scott, Young, and Haynes (1974) found that analogue feedback was superior to binary feedback for learning heart rate speeding but not for learning heart rate slowing. In contrast to those data which support the predictions from Lang's theory, there are several studies which do not support Lang's hypotheses. Four studies have found procedural variables which affect both heart rate speeding and slowing: analogue vs. binary feedback (Colgan, 1977); feedback delay (Williamson & Blanchard, 1979a); and instructing subjects that heart rate is the response to be modified (Blanchard, Scott, Young, & Edmundson, 1974; McCanne & Sandman, 1975). One parameter which has been shown to affect learning a motor skill, distribution of practice, showed no significant effect on learning to increase heart rate (Haynes, Blanchard, & Young, 1977). Overall, the data lend mixed support to Lang's theory. His theory still provides testable hypotheses, i.e. that procedural variables which have been shown to produce a significant effect on learning a motor skill will affect learning to increase heart rate but will not affect learning to decrease heart rate.

Brener's theory of learned heart rate control (1974a, 1974b, 1975, 1977) is part of his general theory of the development of voluntary autonomic control. This theory is

based upon James' (1890) ideomotor theory of voluntary action. Brener's theory focuses largely on the internal processes of developing voluntary heart rate control and proposes that those principles which apply to the learned control of skeletal movements also apply to learned heart rate control. The emphasis placed on feedback loops makes Brener's model related to Adams' (1971, 1976) closed-loop theory of motor learning. The central proposition of Brener's theory states, "that the ability of subjects to discriminate the consequences of their actions is a prerequisite to the development of instructional control over those actions" (Brener, 1974c, p. 585). Much of Brener's theory is devoted to constructing definitions and postulates which make this central proposition testable. Brener proposed that the discrimination of the interoceptive consequences, i.e. internal sensations, of heart rate changes develops through the association of these interoceptive stimuli with a source of exteroceptive (external) feedback which reflects immediate, momentary changes in heart rate. He calls this associative learning process "calibration", and proposed that this process leads to a representation of those interoceptive stimuli associated with changes in heart rate being stored in the subject's memory. This stored memory is termed the "response image". Following sufficient training, this response image can be automatically elicited either by instructions to change the response or by the external

feedback stimulus. Elicitation of this response image then activates the response pattern represented by the image. Therefore, in Brener's theory, development of the response image is essential in learning to control heart rate with or without feedback.

Several hypotheses derived from Brener's theory have been tested (Williamson & Blanchard, 1979c). First, since voluntary control of heart rate requires being able to discriminate changes in heart rate, subjects who are more "aware" of their cardiac functioning should achieve cardiac control more quickly than would subjects who are less "aware". Several tests of this hypothesis have been reported using different behavioral discrimination tasks. One study supported Brener's hypothesis, showing that greater ability to discriminate heart rate was associated with better performance during heart rate biofeedback (McFarland, 1975). A study Brener (1974b) reported found subjects trained to discriminate heart beats were able to achieve better heart rate control than were subjects who received no training. Brener (1977) briefly mentioned a study in which subjects were classified as high or low on cardiac "awareness" on the basis of a heart beat discrimination task. Subjects who scored high in cardiac "awareness" produced significantly larger heart rate increases than did subjects ranked low on "awareness". These two groups did not differ in their ability to produce heart rate decreases. A study using a different

discrimination task did not replicate Brener's (1977) findings (Whitehead, Drescher, Heiman, & Blackwell, 1977). These conflicting results appear to be due partially to the difficulty in adequately assessing cardiac "awareness" (Williamson & Blanchard, 1979c). A recent study (Ross & Brener, 1981) compared training on two different behavioral discrimination tasks. The results indicated that successful learning of a heart rate discrimination task did not assure learning heart rate control in the absence of heart rate biofeedback. Post-hoc classification of subjects according to strategy of solving the discrimination tasks yielded two strategies which were differentially associated with learning to increase heart rate. Ross & Brener interpreted this post-hoc analysis as being generally supportive of Brener's theory. In light of the absence of a relationship between heart rate discrimination and heart rate control combined with the post-hoc nature of the supportive analysis, a definitive statement about the adequacy of Brener's hypothesis concerning cardiac awareness awaits further research.

A second prediction from Brener's theory is that subjects should improve their ability to control heart rate following heart rate biofeedback. This prediction is derived from Brener's postulate that biofeedback "calibrates" the interoceptive cues associated with heart rate and leads to the development of a response image. Thus, after training biofeedback is no longer required for

heart rate control. Pre-feedback and post-feedback phases of heart rate control without feedback must be included to test this prediction. Brener's prediction would be supported if post-feedback heart rate changes were greater than pre-feedback heart rate changes. Six studies have included such phases, four group studies (Bell & Schwartz, 1975; Colgan, 1977; Williamson & Blanchard, 1979a; Lang & Twentyman, 1974) and two multiple single-subject reports (Blanchard, Young, Scott, & Haynes, 1974; Wells, 1973). Five of these studies included heart rate slowing trials and all of these studies confirmed Brener's prediction. For heart rate speeding, only the results of Bell and Schwartz (1975) failed to confirm Brener's prediction. Overall, the majority of the data support Brener's hypothesis regarding learned self-control of heart rate.

One final prediction from Brener's model is derived from the model's emphasis on the necessity of a temporally contiguous association between interoceptive stimuli and exteroceptive feedback for calibration, development of the response image, and development of voluntary control. It would be predicted that a disruption of this temporal contiguity would disrupt the voluntary control of heart rate. One direct test of this prediction has been reported (Williamson & Blanchard, 1979a). This study found that immediate feedback was superior to delayed feedback for both heart rate speeding and slowing. Gatchel (1974) performed an indirect test of this hypothesis by comparing groups

which received immediate beat-by-beat feedback with groups receiving feedback averaged over five or ten interbeat-intervals (the time interval between heart beats). This manipulation of feedback changed both the temporal contiguity and the frequency of information the subject received. Immediate feedback was superior to averaged feedback for heart rate speeding but not for heart rate slowing, partially supporting Brener's prediction. As the most direct test of this hypothesis completely confirmed the model's prediction, this portion of Brener's model is fairly strongly supported.

Overall, Brener's model of voluntary heart rate control has been supported by most of the relevant studies. His model is valuable in that it provides a number of hypotheses which can be tested. However, none of the three leading theoretical models of voluntary heart rate control is comprehensive (Williamson & Blanchard, 1979c). Each theorist has focused on a circumscribed part of the process of learning to control heart rate. There is a need for further theoretical research in order to provide the empirical groundwork for a more comprehensive theory. A common link among the theories of Schwartz, Lang, and Brener is the utilization of portions of motor skills theory. One approach to further research might examine the effect on learned heart rate control of other parameters found in the motor skills literature.

It has been recognized in the motor skills literature

that the informational content of feedback is quite important in learning new tasks (Bilodeau, 1969). Two variables which manipulate this informational content have been shown to influence learning voluntary control of heart rate. Analogue feedback has consistently been shown to be superior to binary feedback in learning heart rate increases. Analogue feedback provides information to the subject about the magnitude of the change produced while binary feedback shows only whether a criterion has been met. Heart rate feedback provided on a beat-by-beat basis is superior to feedback presented less frequently for learning to increase heart rate. That is, more frequent information about performance is superior to less frequent, averaged information. One parameter of the informational content of feedback which has received attention in the motor skills literature is continuous scale transformations. This term refers to systematic changes in the relationship between a subject's response and the feedback that the subject receives regarding that response. For example, Hunt (1964) manipulated the relationship between subjects' responses in a continuous tracking task and the feedback received about those responses. The feedback either magnified error, reduced error, or presented a realistic error signal. It was found that the realistic error signal was associated with greater tracking accuracy. The motor skills literature has demonstrated that continuous scale transformations produce systematic effects in skill learning. Such effects

have been shown for discrete motor responses, e.g. micrometer turning (E. A. Bilodeau, 1953; Noble & Broussard, 1955), and in continuous motor responses, e.g. tracking (Battig, Nagel, & Brogden, 1955; Hartman & Fitts, 1955). One of these studies (Battig, Nagel, & Brogden, 1955) found a curvilinear relationship between errors early in training and the amount by which error was magnified on the tracking display. Using such motor skill tasks, it has been found that the effects of scale transformations are diminished after fifteen to twenty training trials (Bilodeau, 1969).

Recently, an investigation of the effect of a continuous scale transformation on learning to increase heart rate was completed (Williamson, Monguillot, Hutchinson, Jarrell, & Blouin, 1981). By manipulating the calibration of the coupler which converts a subject's electrocardiogram (ECG) to heart rate, it was possible to produce systematic changes in the relationship between heart rate and the feedback which the subject received. Two calibrations were used, producing two different levels of sensitivity of feedback. For a given change in heart rate, the less sensitive feedback condition received exactly one-half as much change in the feedback meter as did those subjects in the more sensitive feedback condition. It was predicted that subjects in the more sensitive feedback group would increase heart rate to a larger degree than would subjects in the less sensitive feedback group, since previous research on voluntary heart rate control indicated

that feedback which provided greater information is generally superior to less informative feedback. Contrary to the prediction, the less sensitive feedback group produced larger heart rate increases.

In retrospect, it appears that there may be an optimal amount of information which can be used by subjects learning to control heart rate. Lang (1974) suggested that such an optimum might be found for learning heart rate control using biofeedback. Lang proposed that immediate feedback of the functioning of a rapidly fluctuating organ could be too rapid for subjects to process. Williamson et al (1981) found significant differences persisting to the 96th trial between the heart rate increases of the two sensitivity conditions. This finding stands in contrast to findings in the motor skills literature concerning the persistence of the effects of continuous scale transformations. Continuous scale transformations affect learning a motor response by slowing the acquisition of the desired response, but all groups eventually approach the same asymptotic performance (Bilodeau, 1969). Significant differences in learning due to continuous scale transformations dissipate by the fifteenth to twentieth trial. The discrepancy in the persistence of continuous scale transformations between the motor skills findings and the learned heart rate control findings highlights the need for a fine-grained examination of methodological variables before adopting any particular learning theory for voluntary heart rate control (Williamson

& Blanchard, 1979c).

Finding that the level of sensitivity of the feedback signal affects the level of learned heart rate increase has potentially far-reaching implications for the study of feedback-assisted learned heart rate control. Brener's theory would predict that the effect of feedback sensitivity would extend to learning to decrease heart rate and to developing self-control of heart rate. Brener's theory would also predict that the effects of feedback sensitivity should be seen in the asymptotic performances of groups learning to increase or decrease heart rate with feedback of varying sensitivity. Lang's model would predict that the effect of feedback sensitivity would be limited to learning to increase heart rate. Studying the effects of feedback sensitivity on learning to decrease heart rate and developing self-control of heart rate would have implications for the research and applied uses of heart rate biofeedback as well as for Brener and Lang's theories. Previously published studies have rarely specified the sensitivity of the feedback signal used. The previously unrecognized effect of feedback sensitivity on learning to increase heart rate may account for some of the contradictory findings in that literature. Also, the contradictory findings concerning parameters which affect heart rate slowing may be due in part to unrecognized feedback sensitivity effects. Examining the effects of feedback sensitivity on learning to decrease heart rate

might isolate one source of variability in the heart rate slowing literature. As the development of self-control of heart rate would be desirable in any clinical use of feedback-assisted learned heart rate control, examining the effect of feedback sensitivity on learned self-control of heart rate would have implications for the applied uses of heart rate biofeedback.

Problem

The preceding discussion established that an examination of the effects of feedback sensitivity on learning to decrease heart rate and learning self-control of heart rate would have implications for the theoretical models of Brener and Lang as well as for applied and methodological issues in feedback-assisted learned heart rate control. The present study was designed to extend the research concerning the effects of feedback sensitivity upon learned heart rate control. More specifically, the study examined the effects of feedback sensitivity on: (a) learning to decrease heart rate; (b) developing self-control of heart rate; and (c) the asymptotic performance of learned heart rate increases and decreases. The present study also attempted to replicate the effect of feedback sensitivity on learning to increase heart rate.

The design of this study, as illustrated in Figure 1, included one between-group factor and three repeated measures. The between-group factor was sensitivity of feedback. Two levels of feedback sensitivity were included,

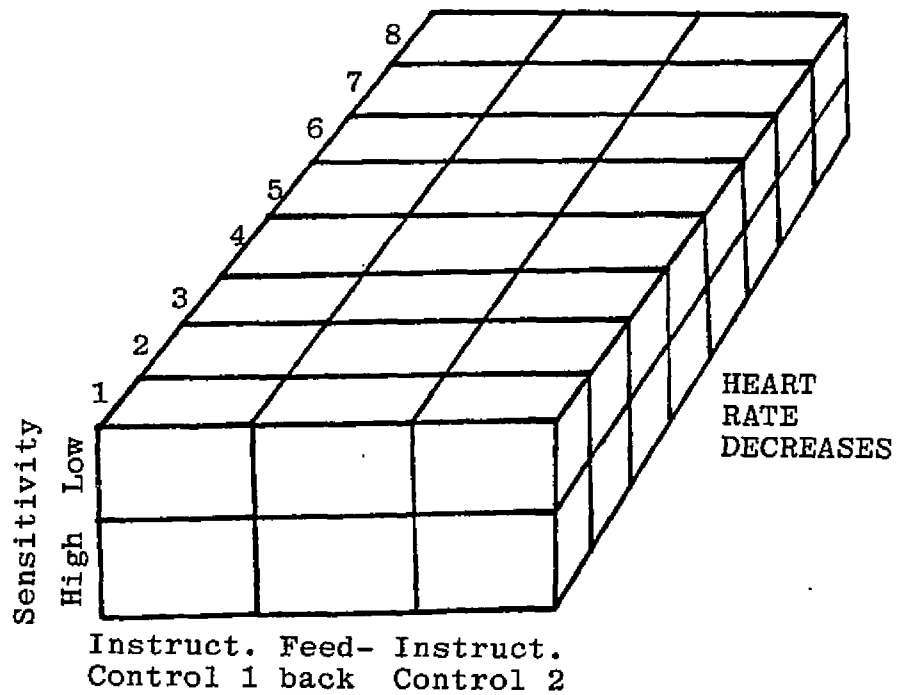
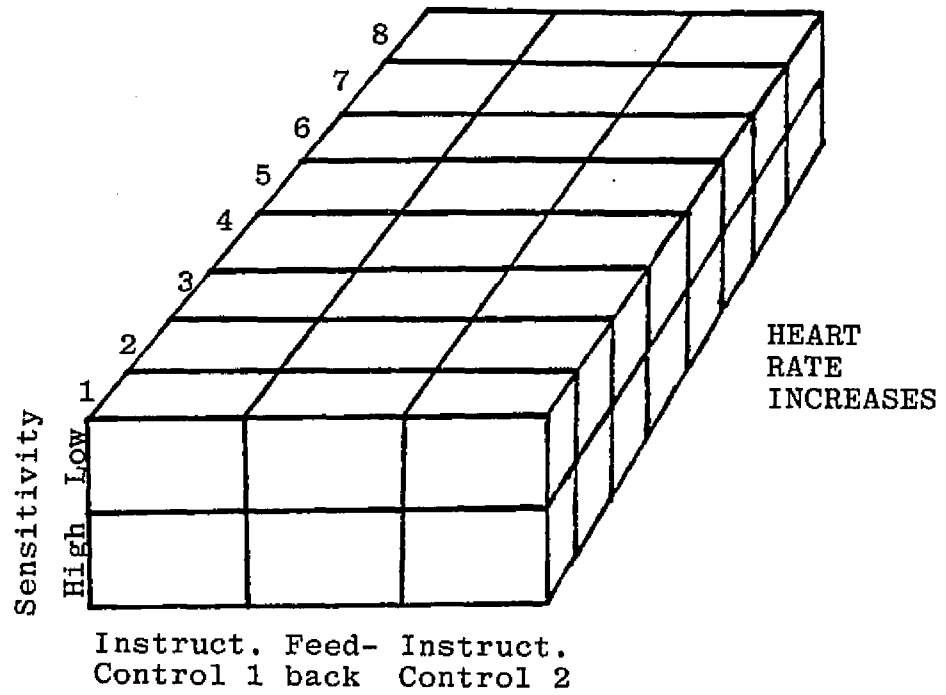


Figure 1. Experimental design showing the four factors and the levels of each factor.

high sensitivity and low sensitivity. The high sensitivity condition received a 1 cm change in the feedback display for an 8.57 beat-per-minute change in momentary heart rate. The low sensitivity condition received a 1 cm change on the feedback display for a 17.14 beat-per-minute (BPM) change in momentary heart rate. The first repeated measure was direction of heart rate control, i.e. Increase or Decrease. The second repeated measure was within-session phases of: (1) pre-feedback Instructional Control in which subjects were instructed to increase or decrease heart rate without the aid of feedback; (2) heart rate Feedback with instructions to increase or decrease heart rate; (3) a post-feedback Instructional Control phase identical to the pre-feedback Instructional Control phase. The third repeated measure was extended training. In order to assess the learning of heart rate control over time, subjects received eight sessions of training.

Four hypotheses were tested in the present study. The first hypothesis represented an attempt to replicate the findings of Williamson et al (1981) regarding the effect of sensitivity of heart rate biofeedback on learning to speed heart rate. Hypothesis 1 was formally stated as:

Hypothesis 1: If there is an optimal degree of sensitivity of heart rate biofeedback above which increases in feedback sensitivity will produce decrements in learning to increase heart rate, then the effect of sensitivity of feedback should

result in the ordering of groups as follows for heart rate speeding:

low sensitivity \succ high sensitivity

The final three hypotheses were derived from the theories of Brener and Lang. These hypotheses will be grouped according to theorist, with Brener's hypotheses being considered first. Brener's model proposes that variables which affect the establishment of control over a bodily response are mediated through effects on the response image. Brener's model does not differentiate learning to increase heart rate from learning to decrease heart rate, therefore a variable which affects learning to increase heart rate should equivalently affect learning to decrease heart rate via the response image. Hypothesis 2 was formally stated as follows:

Hypothesis 2: If an optimal degree of sensitivity exists above which increases in feedback sensitivity will produce decrements in learning to control heart rate and if learning to decrease heart rate is affected similarly by variables which affect learning to increase heart rate, then the effect of feedback sensitivity should result in the following ordering of groups for learning to slow heart rate when considering the absolute value of heart rate change:

low sensitivity \succ high sensitivity

Brener's model considers development of an optimal

response image necessary for learning to produce the largest possible change in heart rate. Therefore, variables which can have a detrimental effect on learning to control heart rate should result in less than optimal response images. As a result, the asymptotic performance of the high sensitivity feedback group should not be as large as the asymptotic performance of the low sensitivity feedback group. Hypothesis 3 was formally stated as:

Hypothesis 3: If an optimal degree of feedback sensitivity exists above which increases in feedback sensitivity will produce decrements in learning to control heart rate, then in the final session of training, the performance of the sensitivity groups should be ordered as follows for both increasing and decreasing heart rate when considering the absolute value of heart rate change:

low sensitivity \succ high sensitivity

The final prediction from Brener's model dealt with learning self-control of heart rate, i.e. without feedback. Brener's model proposes that the development of self-control relies on establishing a response image for the response to be controlled. Therefore, variables which affect the response image should show similar effects on learned self-control. Improved self-control was defined as producing significantly larger heart rate changes during post-feedback instructional control phases as compared to

the initial instructional control phase of session 1.
Hypothesis 4 was formally stated as:

Hypothesis 4: If there is an optimal level of feedback sensitivity above which increases in feedback sensitivity lead to decrements in learned self-control of heart rate, then for both increasing and decreasing heart rate, a comparison of the absolute value of the two sensitivity groups' performances on the initial pre-feedback Instructional Control phase and later post-feedback Instructional Control phases should find:

self-control (post-feedback Instructional Control—pre-feedback Instructional Control) of the low sensitivity group \geq self-control of the high sensitivity group

The final hypothesis was derived from Lang's theory. Lang's model makes a distinction between learning to increase heart rate and learning to decrease heart rate. This model proposes that variables which affect learning motor skills will affect learning to speed heart rate but will not affect learning to slow heart rate. This prediction was opposite to that of Hypothesis 2 and will be stated as Hypothesis 2(a).

Hypothesis 2(a): If an optimal degree of sensitivity exists above which increases in feedback sensitivity will produce decrements in

learning to control heart rate and if learning to slow heart rate is not affected by variables which affect learning to increase heart rate and learning motor skills, then the effect of feedback sensitivity should result in the following ordering of groups for learning to slow heart rate:

low sensitivity = high sensitivity

METHOD

Subjects

Twenty undergraduate students from introductory psychology courses at Louisiana State University were randomly assigned to two groups which received heart rate feedback of two different sensitivities. All subjects denied using medications and denied any history of heart disease. Equal numbers of males and females were assigned to each group. Subjects ranged in age from 18 to 23 years. All subjects received extra course credit for participating in the experiment.

Five undergraduate students participated in a brief study of the visibility of changes in the feedback meter. Three males and two females volunteered for this study. All subjects reported that their vision was normal without corrective lenses. None of these five students participated in the heart rate training sessions.

Apparatus

All physiological measures were recorded on a Beckman R-411 polygraph. The electrocardiogram (ECG) and temperature changes caused by breathing expiration-inspiration cycles were recorded using two Beckman voltage/pulse couplers (9853A). Electromyographic activity (EMG) was recorded using the direct signal of a Beckman EMG

coupler (9851). Heart rate was recorded and the feedback meter reflected the output from the cardiograph channel (Beckman coupler 9857B). Heart rate biofeedback was provided using a Med Associates needle feedback meter (ANL-920) with a full-scale meter deflection of 7 cm. The feedback meter was labeled to indicate the direction of heart rate change indicated by movement of the feedback meter needle. Heart beats were automatically counted using a Med Associates threshold comparator and print out counter. The timing of all trials and the presentation of all within-session instructional stimuli, i.e. red, green, and yellow lights, was automatically controlled by Med Associates solid-state logic and programming equipment. Subjects were seated in a cushioned chair in front of the feedback meter and the instructional lights. The instructional lights were part of a Med Associates three-color display (DIG-935). The three instructional lights were labeled to remind the subject of the instructions related to it, e.g. green-raise heart rate. Standard plate electrodes were placed on the subject's legs and left arm for the recordings of ECG and heart rate. Three silver/silver chloride electrodes, one ground electrode and two active electrodes, were positioned 4.2 cm apart on the subject's forehead for the recording of frontal electromyogram (EMG). These electrodes were mounted in an adjustable headband and the ground electrode was placed approximately 2.5 cm above the midline of the nose, placing

each active electrode the same distance above each eyebrow. A Yellow-Springs thermistor, positioned beneath one nostril, was used for the recording of respiration inspiration-expiration cycles.

Feedback sensitivity was manipulated by calibrating the tachograph channel so that a 60 beat per minute (BPM) change in heart rate produced either a 10 cm polygraph pen deflection for the high sensitivity condition (Condition A) or a 5 cm polygraph pen deflection for the low sensitivity condition (Condition B). A heart rate of 90 BPM had equivalent outputs for each sensitivity. As a result of the tachograph calibration, condition A provided a heart rate signal of relatively high sensitivity with a momentary heart rate change of 8.57 beats-per-minute (BPM) producing a feedback meter needle deflection of 1 cm. In contrast, condition B provided feedback which was exactly one-half as sensitive as condition A, i.e. a momentary heart rate change of 17.14 BPM produced a needle deflection of 1 cm.

Procedure

Visibility of Feedback Needle Movement Study

A brief study of the visibility of movements of the feedback meter needle was performed to assess the degree of heart rate change required to produce discriminable deflection of the feedback needle. Each of the five subjects who participated in this study were seated in front of the feedback meter at the same distance as the subjects involved in heart rate training (approximately .75 m). Six

levels of needle movement were evaluated: (1) .125mm, (2) .25 mm, (3) .5mm, (4) 1.25mm, (5) 2.5 mm, (6) no movement. Each subject viewed five trials at each of these six levels, with the order of presentation of the thirty trials being randomized. Trials were presented every ten seconds. Each trial consisted of a pulsed (approximately .5 sec) needle movement followed by an inquiry of the subject as to whether the needle had moved. Following each subject's participation, the purpose of this study was explained and all questions were answered.

Effect of Feedback Sensitivity on Heart Rate Control Study

Each of the twenty experimental subjects in this study received eight one-hour sessions of heart rate biofeedback scheduled during a sixteen-day period. Each session consisted of the same four within-session phases. These phases are shown in Figure 2. All trial lengths were $1\frac{1}{2}$ minutes, and each trial alternated with a $1\frac{1}{2}$ minute rest period.

After attaching the electrodes in Session 1, subjects were asked to read the set of written instructions presented in the next section. Before beginning the other seven sessions, subjects were asked if they had any questions concerning the nature of the experimental task. Questions covered in the written instructions were answered verbally. During the remainder of the adaptation phase, subjects were

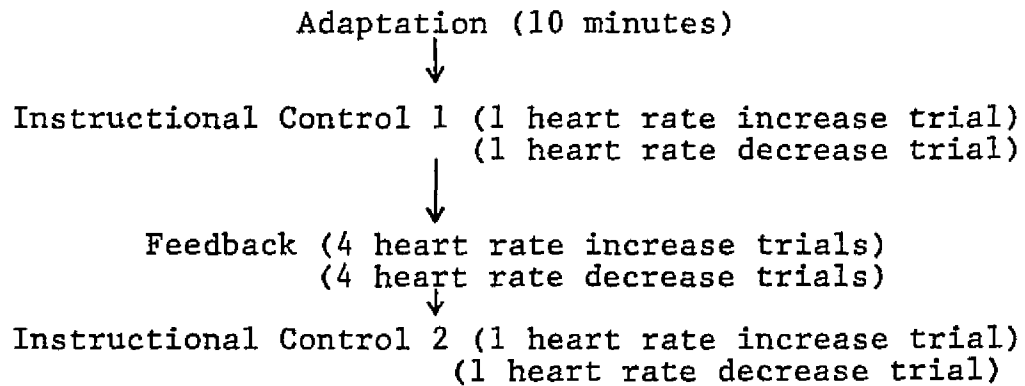


Figure 2. The four within-session phases

instructed to sit quietly so that any unnecessary physiological arousal due to walking, etc. would diminish before beginning the session.

The feedback display was not functional during the Instructional Control phases. Heart rate increase trials were signalled by the green light. Heart rate decrease trials were signalled by the yellow light. The rest periods, which alternated with all other trials, were signalled by the red light. All instructional lights were illuminated for the duration of the appropriate trial.

The meter facing the subjects provided proportional heart rate feedback during the feedback trials. Feedback appeared as a meter needle which moved to the right when heart rate was increasing and moved to the left when heart rate was decreasing. Feedback was not presented during rest trials. The order of heart rate increase and decrease trials was counterbalanced for all groups and for all within-session phases. The two conditions of feedback sensitivity were created by calibrating the tachograph as

described earlier. Subjects were not informed of the differing feedback sensitivities until after their participation in the experiment had finished.

Each subject was interviewed using a structured questionnaire following his or her final session. The questionnaire consisted of five questions. Three questions designed to assess whether subjects had been aware of the differences in feedback sensitivity were alternated with two filler questions. The three questions concerning awareness of the manipulation ranged from ambiguous to direct (Aronson & Carlsmith, 1968). The first question asked, "Was there any aspect of the experiment that seemed unusual to you?" The second question asked, "Did anything about the feedback display seem unusual to you?" The third question was more direct, asking, "Do you believe that your feedback needle moved differently from the feedback presented to the other subjects?" Answers to each of these questions were recorded verbatim unless the answer was long and unrelated to the sensitivity manipulation. In such cases, the responses were summarized.

Instructions

Before each session, each subject was instructed to read the following set of instructions:

This experiment is designed to determine how well you can learn to control your heart rate. Many previous experiments have found that this task can be learned. Apparently, there is no one

way to accomplish this task since different subjects learn to control their heart rates by different means. You may use whatever strategy you find works best, with the exceptions of changing your breathing pattern, e.g. holding your breath, or increasing muscular tension, e.g. clenching your fists. The meter in front of you will inform you of your heart rate by the position of the needle. If the needle moves to the right, that means your heart rate is increasing. If the needle moves to the left, that means your heart rate is decreasing. The farther the needle moves, the more you are changing your heart rate. Sometimes the needle may not move immediately when you first attempt to control your heart rate during a trial.

After the experimenter leaves the room, a ten-minute adaptation period will follow. During this period, just sit quietly. Refrain from moving your arms, legs, etc. as much as possible during this adaptation phase as well as during all other phases of the experiment. Following the adaptation phase, either the green light or yellow light will be illuminated for $1\frac{1}{2}$ minutes. The feedback display will not operate during the first two trials of each session. During these no-feedback trials, attempt to change your heart

rate in the direction indicated by the light which is illuminated, i.e. green light - raise heart rate; yellow light - lower heart rate. Following the first no-feedback trial and following all other trials, the red (rest) light will be illuminated for 1½ minutes. During this period, simply rest and sit quietly. Do not attempt to practice heart rate control during this or any other rest period. After the rest period ends, the second no-feedback trial will begin. Once again, attempt to change your heart rate in the direction indicated by the illuminated light. Following the two no-feedback trials, feedback trials will begin. During these trials, attempt to use the feedback to change your heart rate in the direction indicated by the light which is illuminated. Following feedback trials, you will be given two more no-feedback trials. The same instructions which apply to the initial no-feedback trials apply to these no-feedback trials.

The same instructions will apply for all of your experimental sessions. You will be given an opportunity to look over these instructions before each session.

Remember, you should try to alter your heart rate as much as possible and for as long as

possible during the heart rate raising and lowering trials. During the rest trials, sit quietly and do not alter your heart rate. If you have any questions, feel free to ask the experimenter. Thank you for your participation.

Data Reduction and Analysis

Data from the feedback needle movement study were summed across all subjects for each of the six levels of needle movement.

Data for each of the physiological responses of the feedback sensitivity study, i.e. heart rate, frontal EMG, and respiration were computed from all artifact-free periods of each session. Two portions of data were lost due to procedural problems. All data from session seven for one subject in the low sensitivity condition were lost due to a clerical error. The respiration data from session three for another subject in the low sensitivity condition were lost due to equipment failure. These periods of missing data were managed by eliminating them from the statistical analysis and adjusting the degrees of freedom accordingly. Heart rate was computed by dividing the total number of heart beats by the total time of each trial period. Frontal EMG was quantified by having the direct EMG signal converted into digital data using a Med Associates Analogue to Digital Converter (ANL940). The digital information was later transformed into appropriate units, i.e. microvolt-seconds, and averaged across trials to produce trial means.

Respiration rate was computed by hand counting the number of pen displacements caused by an inspiration-expiration cycle and then dividing by the trial time. A second judge scored a random sampling of ten percent of the respiration data in order to provide a reliability check. Spearman rank correlation coefficients were computed for each of the 16 sessions which had respiration data scored by the second judge. These coefficients ranged in value from .98 to .89, with a median value of .92.

Statistical analysis of these data was performed using difference scores obtained by subtracting the scores obtained from the last 60 sec. of the preceding rest period from the scores of each trial. Data from the first 30 sec. of the 90 sec. rest periods were discarded since subjects were requested to move only during those time periods. Data from the trials within the Feedback phase were averaged for each session as no predictions were made about individual trials within the Feedback phase.

The heart rate data were analyzed to detect overall differences across conditions using two separate analyses of variance (ANOVA). The first ANOVA was a $2 \times 2 \times 3 \times 8$ analysis with one between-group factor, feedback sensitivity, and three repeated measures, i.e. Direction, Within-session Phases, and Sessions. The second ANOVA was employed with only the data from Session 8 so that Hypothesis 3 could be analyzed separately. Therefore, it consisted of a $2 \times 2 \times 3$ ANOVA with one between-group

factor, feedback sensitivity, and two repeated measures, Direction and Within-session Phases. Planned comparisons of the different cell means were carried out using orthogonal comparisons. The alpha level for acceptable statistical significance was $p < .05$.

The EMG and respiration rate data were analyzed using a $2 \times 2 \times 3 \times 8$ ANOVA that is identical in form to the first ANOVA for heart rate described above. Since no planned comparisons were conceptualized for these data, any post-hoc comparisons carried out used Scheffe's test with the alpha level set at $p < .05$.

RESULTS

The Results section is divided into several sections. First, an initial consideration of the visibility of feedback needle movement is discussed. The next section addresses the issue of changes in heart rate across the series of rest trials, which are of importance in interpreting the later analyses of heart rate data. The analyses of the heart rate data are presented next, with these analyses being of primary importance in the evaluation of the experimental hypotheses. Subjects' awareness of the experimental manipulation is then examined, followed by analyses of EMG and respiration data.

Perception of Feedback Needle Movement

The results of this study are presented in Table 1. Examination of this table shows that all subjects were able to perceive meter needle movements of 1.25mm and greater with 100% reliability. A feedback needle movement of 1.25mm was approximately equivalent to a .22 beats-per-minute (BPM) change in momentary heart rate for the low sensitivity condition and approximately equivalent to a .11 BPM change for the high sensitivity condition. These data indicate that the smallest change in the feedback needle which all subjects in the low sensitivity condition would have been able to reliably perceive was sufficiently small so as to

provide a reasonably responsive indicator of momentary heart rate change.

Needle Movement	"Moved"	"Didn't Move"
.125mm	10%	90%
.25mm	26%	74%
.5mm	54%	46%
1.25mm	100%	0
2.5mm	100%	0
-0-	4%	96%

Table 1. Data summary of feedback meter needle movement perception study

Heart Rate Data

A 2 x 2 x 3 x 8 ANOVA was performed upon rest trial heart rates to evaluate possible differences in resting heart rates. The only significant effect in this analysis was a Condition x Session x Direction interaction, $F(7,429) = 2.311$, $p < .05$. Post-hoc comparisons among these data using Scheffe's criteria indicated that the means of Session 2 differed significantly. The mean resting heart rates during Session 2 were significantly greater for the low sensitivity condition (increase trials, $\bar{X}=75.97$ BPM; decrease trials, $\bar{X}=76.37$ BPM) than for the high sensitivity condition (increase trials, $\bar{X}=69.41$ BPM; decrease trials, $\bar{X}=68.84$ BPM), with t' values ranging between 12.50 and 14.34, $p .01$. No other significant differences were found among rest trial heart rate means during any other session.

The appearance of this difference in resting heart rates in the second session necessitated an examination of the possible effect of this difference on further statistical analyses. The second session difference in resting heart rate might have been expected to influence the magnitude of heart rate change produced. For example, the Law of Initial Values would predict that a decline in resting heart rate would reduce the range of heart rate decreases which could be produced while increasing the range of heart rate increases which could be produced. Inspection of Figure 3, which presents the heart rate change data across by condition and direction, shows the change in heart rate effects in session 2. While little change in heart rate decrease performance is seen for either condition in session 2, a marked change in heart rate increase performance is seen. The high sensitivity condition showed a marked decline in heart rate acceleration performance, while the low sensitivity condition showed a marked improvement in heart rate acceleration performance. As the heart rate increase results were the only heart rate change data to show a marked shift in session 2, correlational analyses of the relationship between resting heart rates and heart rate increases were performed for this session's data to examine what effect the difference in resting heart rate may have had on heart rate increase performance.

Pearson product moment correlation coefficients computed on resting heart rates and heart rate increases for

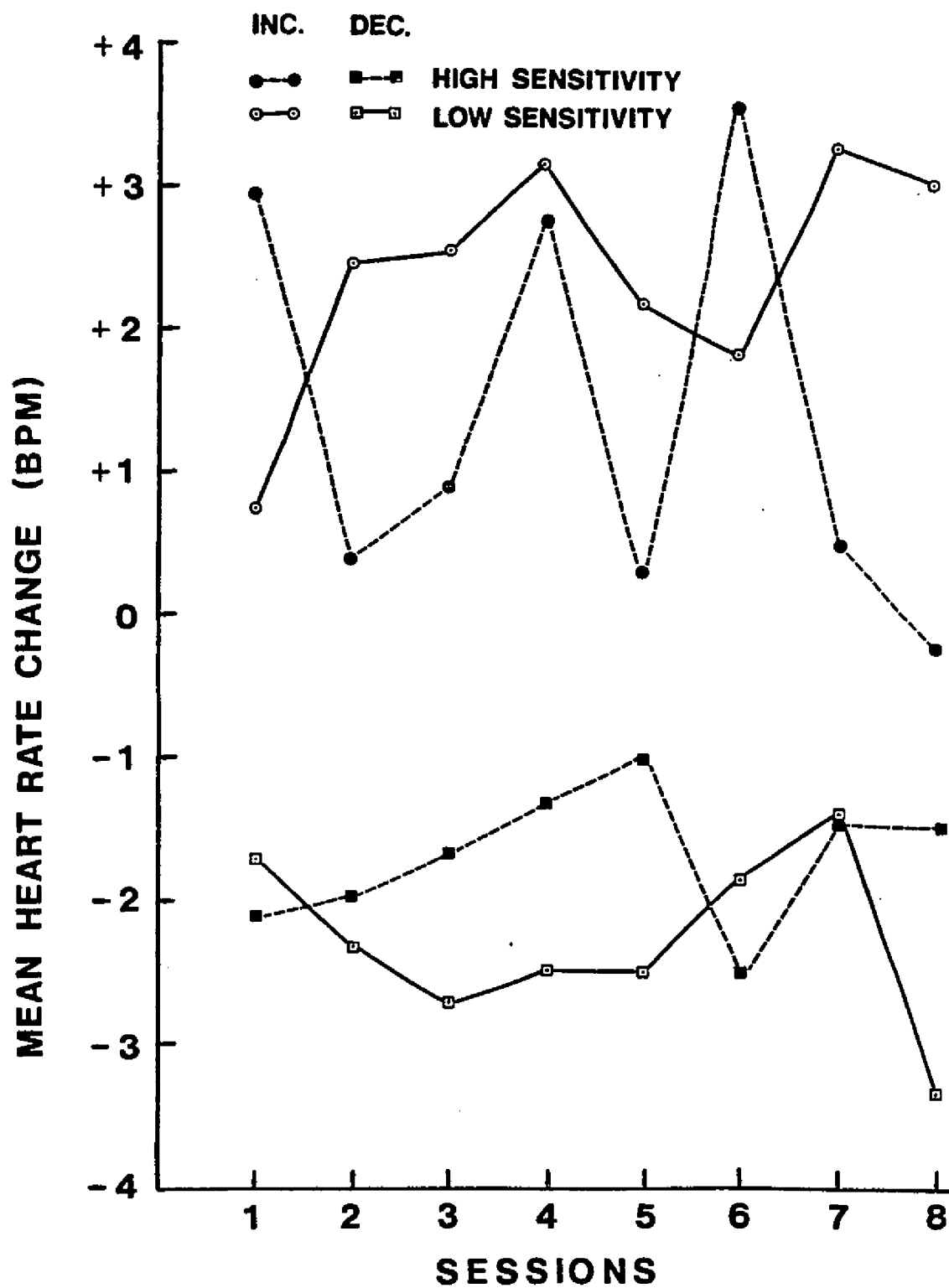


Figure 3. Mean heart rate increases (INC.) and decreases (DEC.) for each condition by session.

session 2 yielded nonsignificant coefficients which did not differ significantly between conditions (high sensitivity, $r = -.185$; low sensitivity, $r = -.044$). These correlation coefficients indicate that the change in heart rate increase performance seen in session 2 was unrelated to the variation in resting heart rates during this session. Based upon these results, it was concluded that the variation in resting heart rate during session 2 was unrelated to the magnitude of heart rate change produced and that evaluation of treatment effects would proceed on difference scores as planned.

The evaluation of treatment effects was completed using difference scores. A $2 \times 2 \times 3 \times 8$ ANOVA was performed to evaluate Hypotheses 1, 2, and 4. This analysis indicated that the difference between trials in opposing Directions, increase and decrease, was significant, $F(1,429) = 198.72$, $p < .01$. This effect indicates that subjects were able to change their heart rates significantly. A significant Condition \times Direction interaction was also found in this analysis, $F(1,429) = 7.607$, $p < .01$. This interaction is illustrated in Figure 4 and relates to Hypotheses 1 and 2. Planned comparisons among these means showed that subjects in the low sensitivity condition produced significantly larger heart rate increases than did subjects in the high sensitivity condition, $t(476) = 2.456$, $p < .05$. No significant difference was found between the two conditions for mean heart rate deceleration.

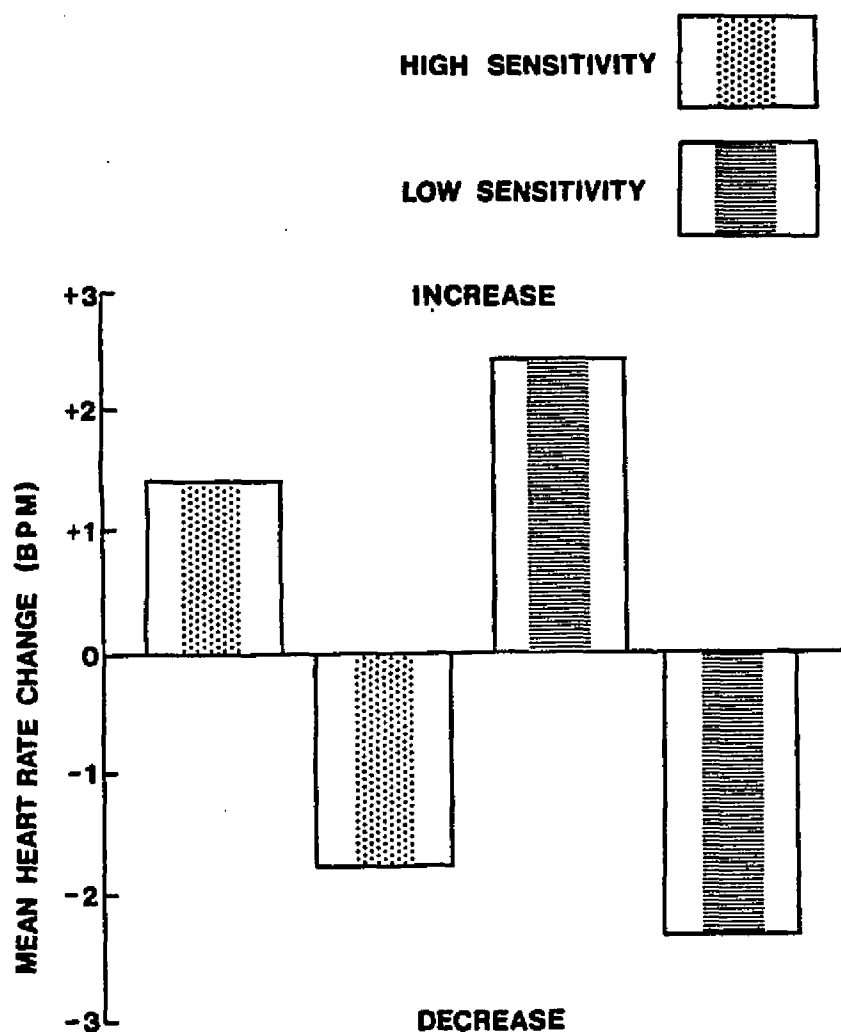


Figure 4. Mean heart rate increases and decreases for each condition averaged across all sessions.

The $2 \times 2 \times 3 \times 8$ ANOVA performed on heart rate difference scores also yielded a significant Condition \times Direction \times Session effect, $F(7,429) = 2.853$, $p < .01$. These data are illustrated in Figure 3. This effect indicates that differences between the performance of the two conditions varied across the eight sessions. As no comparisons were planned for these data, post-hoc comparisons were made using Scheffe's criteria. No post-hoc comparisons were significant under these criteria. No main effect or interaction involving the Phase factor was found in the $2 \times 2 \times 3 \times 8$ ANOVA on heart rate difference scores. This result bears upon Hypothesis 4, as the predicted relationships among the within-session phases was expected to be reflected in the Phase factor.

A $2 \times 2 \times 3$ ANOVA was performed on the heart rate difference scores of the eighth session in order to evaluate the asymptotic effects of training. A significant main effect for Direction of heart rate change was found, $F(1,54) = 22.94$, $p < .01$. A significant Condition \times Direction interaction was also found, $F(1,54) = 8.78$, $p < .01$. This interaction is of importance in evaluating Hypothesis 3 and planned post-hoc comparisons were performed on these data. These comparisons indicated that the low sensitivity condition produced significantly larger heart rate increases ($\bar{X} = 2.97$ BPM) than did the high sensitivity condition ($\bar{X} = -.23$ BPM), $t(54) = 3.92$, $p < .05$. The comparison of conditions for heart rate decreases failed to

reach significance.

Awareness of Feedback Sensitivity Manipulation

The first two post-experimental questions pertaining to the manipulation of feedback sensitivity were purposefully ambiguous. No subject answered these questions in terms of a manipulation of sensitivity of feedback. The third question asked directly whether the subject believed their feedback needle moved differently from those used with other subjects. Two subjects in each condition answered affirmatively. None of these four subjects were able to identify the manipulation more than affirming the suggestion that the feedback needle moved differently for them. The equal number of affirmative respondents to this third question in each condition and the lack of specificity of these subjects' knowledge about how the feedback needle moved differently indicate that "awareness" of the manipulation of feedback sensitivity could not explain differences in heart rate changes between conditions.

EMG Data

The $2 \times 2 \times 3 \times 8$ ANOVA performed on the EMG difference scores yielded two significant effects. A significant main effect for Direction indicated that subjects significantly varied their frontal muscle tension according to the direction on heart rate change, $F(1,429) = 22.85$, $p < .01$. Mean EMG difference scores equaled $+ .24 \mu\text{V-s}$ for increase trials and equaled $-.59 \mu\text{V-s}$ for decrease trials. A significant Session x Phase interaction was also found,

$F(14,286) = 1.72, p < .05$. A generally increasing trend in EMG was seen across sessions. Post-hoc comparisons using Scheffe's criteria failed to find significant differences among the mean values of Phases across Sessions. Correlational analysis of the overall relationship between EMG change and heart rate change yielded significant correlation coefficients for both heart rate increases ($r = .235, p < .01$) and heart rate decreases ($r = .327, p < .01$). The most significant aspect of the ANOVA is the absence of any effect involving the Condition factor. As a result, the difference in heart rate change across Conditions cannot be explained by changes in EMG levels.

Respiration Rate Data

The $2 \times 2 \times 3 \times 8$ ANOVA performed on the respiration difference scores yielded two significant effects. A significant main effect for Phase indicated that subjects significantly varied their respiration across the different phases, $F(2,284) = 8.12, p < .01$. Changes in respiration rate were positive across the three phases with larger changes being produced in the final two phases of each session. The mean changes were: Instructional Control 1 respiration rate change = $+.145$ respiration cycles; Feedback respiration rate change = $+.72$ respiration cycles; Instructional control 2 respiration rate change = $+.76$ respiration cycles. Post-hoc testing using Scheffe's criteria showed that the differences between these means were not statistically significant.

A significant main effect for Direction was also found in the ANOVA on the respiration difference scores, $F(1,426) = 118.20$, $p < .01$. Post-hoc testing using Scheffe's criteria indicated that subjects significantly changed their respiration rate between heart rate increase trials ($\bar{X} = 1.63$ respiration cycles) and heart rate decrease trials ($\bar{X} = -.55$ respiration cycles). Correlational analysis of the relationship between heart rate change and respiration rate change across all sessions yielded a significant positive relationship for heart rate increases ($r = .188$, $p < .01$) but failed to show a significant relationship for heart rate decreases ($r = -.001$). The most significant finding in the respiration rate data was the absence of any effect involving the Condition factor. As a result, differences between the two conditions could not be explained in terms of differential manipulation of respiration rate as a function of the experimental conditions.

DISCUSSION

The results of this experiment clearly demonstrate the effect of feedback sensitivity on learning to increase heart rate. The low sensitivity condition showed larger magnitude mean heart rate increases than did the high sensitivity condition across all eight sessions and in session eight. These results replicate the earlier findings of Williamson, et al (1981). This effect of feedback sensitivity was not seen in heart rate decrease performance, as no significant differences were found between the sensitivity conditions in heart rate decrease performance. Also, no effect of feedback sensitivity on heart rate performance was found across the feedback and no-feedback phases of the study.

The heart rate change data shows interesting variability given the above findings. While the low sensitivity condition produced larger overall heart rate increases, the high sensitivity condition showed the largest mean session increase in session six. This sixth session heart rate increase was the largest mean increase in any session across conditions as seen in Figure 3. The high sensitivity condition showed marked variability in heart rate increase performance, producing session means greater than +2.5 BPM in sessions one, four, and six while producing

means of less than +1.0 BPM in the other five sessions. The low sensitivity condition did not demonstrate such remarkable variability in heart rate increase performance, and neither condition showed such variability in heart rate decrease performance.

Structured debriefing interviews established that neither condition had significant knowledge of the experimental manipulation, allowing this issue to be dismissed as a possible explanation of the results found. Measurements of changes in respiration rate and frontal EMG also showed no significant difference between conditions. Therefore, changes in these somatic responses appear to be unlikely explanations for the differences between the sensitivity conditions.

The following sub-sections will review the experimental hypotheses with reference to the results presented above. These sub-sections will be followed by a concluding section which will review the theoretical implications of the present study and attempt to explain the present findings with the available theory.

Hypothesis 1

Hypothesis 1 predicted that the low feedback sensitivity group would produce larger magnitude heart rate increases than would the high feedback sensitivity group. This prediction was based upon the findings of Williamson, et al (1981) in a similar study. The present study confirmed this prediction by finding the low sensitivity

group's heart rate increases across the eight sessions to be significantly greater than those of the high sensitivity feedback group.

Hypotheses 2 and 2(a)

Hypothesis 2 was derived from Brener's model and this hypothesis predicted heart rate decrease performance. Brener's model predicts that variables which affect heart rate increases would also affect heart rate decreases through effects on developing the response image. Hypothesis 2 predicted that the low feedback sensitivity group would produce larger magnitude heart rate decreases than would the high feedback sensitivity group.

Hypothesis 2(a) was opposite to Hypothesis 2 and was derived from Lang's model. Lang's model predicts that variables which affect the acquisition of motor skills, such as feedback manipulations, would affect learning to increase heart rate but would not affect learning to decrease heart rate. Hypothesis 2(a) predicted that the low feedback sensitivity group would produce heart rate decreases which were equivalent to those produced by the high sensitivity group.

The present study found no significant difference between the average heart rate decrease performances of the two sensitivity conditions across all sessions. These findings support Hypothesis 2(a), the prediction derived from Lang's model.

Hypothesis 3

Hypothesis 3 was derived from Brener's model and this hypothesis dealt with final session performance. Brener's model states that development of an optimal response image is necessary for learning to produce the largest heart rate changes possible. Variables which have a detrimental effect on learning to control heart rate should affect asymptotic performance detrimentally by producing a less than optimal response image. Hypothesis 3 predicted that in the final session, the low feedback sensitivity condition would produce larger magnitude heart rate increases and decreases than would the high feedback sensitivity condition. This hypothesis was partially confirmed. The predicted relationship was found for heart rate increases. No significant difference between the sensitivity conditions was found for heart rate decreases.

Hypothesis 4

Hypothesis 4 was derived from Brener's model and dealt with differences in heart rate change performance across the within-session phases. Brener's model predicts that development of self-control of heart rate, i.e., producing changes without feedback present, depends upon establishing a response image for the response to be controlled. Decrements in the development of a response image should produce decrements in self-control. Hypothesis 4 predicted that for both heart rate increases and decreases, the difference between the initial pre-feedback Instructional

Control phase and the later post-feedback Instructional Control phases would be larger for the low sensitivity condition than for the high feedback sensitivity condition. This hypothesis was not confirmed for either heart rate increases or decreases.

Integration and Conclusion

Brener's model of learned heart rate control (1974a, 1974b, 1975, 1977) received only partial support from the findings of this study. Three hypotheses were derived from his model, Hypotheses 2, 3, and 4. Hypothesis 3 was partially confirmed, showing the predicted relationship between conditions in the eighth session for heart rate increases but not decreases. The other two hypotheses were not confirmed. All of the experimental hypotheses derived from Brener's model relied upon the proposed necessity of developing the response image for the response to be learned.

Brener's model does not distinguish between different responses to be learned. Hypothesis 2 was derived from this portion of Brener's model, predicting that learning to decrease heart rate would be affected in the same way that learning to increase heart rate was affected. As this hypothesis was not confirmed, this part of Brener's model is called into question. The evidence concerning the similarity of the modification of heart rate increases and decreases will be reviewed when the hypothesis derived from Lang's model is considered. The data from the present

study indicate that Brener's model needs to be examined closely in its failure to distinguish between different responses to be learned.

Hypothesis 3 was based upon a prediction from Brener's model that an optimal response image would be necessary for optimal heart rate change performance and that development of an optimal response image would require optimal feedback. It was predicted that the low sensitivity condition would show better heart rate increase and decrease performances in the eighth session than would the high sensitivity condition. The predicted performance was found for heart rate increases; no difference between the conditions was found, for heart rate decreases. The finding of a significantly greater heart rate increase in the eighth session for the low sensitivity group is consistent with the low sensitivity group's superior heart rate increase performance across the experiment. This finding supports the prediction from Brener's model, i.e. that optimal performance requires optimal feedback. The failure to find the predicted relationship for heart rate decreases once again calls into question Brener's failure to distinguish between different responses to be learned.

Hypothesis 4 was based upon that portion of Brener's model which proposes that once the response image for a particular response is established, the learned response should be capable of being produced in the absence of feedback. Hypothesis 4 predicted that the low sensitivity

condition would show better self-control of heart rate increases and decreases than would the high sensitivity condition. Self-control was defined as the difference in heart rate change performance between the initial pre-feedback Instructional Control phase and post-feedback Instructional Control phases. The present study failed to find significant differences in self-control of heart rate changes across conditions. Finding that feedback sensitivity significantly affected heart rate increases across the whole experiment but that this difference was not seen across non-feedback phases raises questions about the validity of the self-control component of Brener's model. The self-control portion of Brener's model had been supported by five out of six pertinent studies (Colgan, 1977; Williamson & Blanchard, 1979a; Lang & Twentyman, 1974; Blanchard, et al, 1974; Wells, 1973). The one previously non-supportive study (Bell & Schwartz, 1975) is now joined by the non-supportive results of the present study. These findings, which do not support the self-control component of Brener's model, indicate that a further examination of this portion of Brener's model is in order.

Overall, more questions about Brener's model have been raised than have been answered. The findings of the present study partially support that portion of Brener's model which proposes that optimal feedback is required for learning optimal performance in heart rate change. Predictions from Brener's model regarding the similarity in learning to

modify different physiological responses and self-control of heart rate were not supported and these parts of Brener's model deserve further examination.

One hypothesis of the present study, Hypothesis 2(a) was derived from Lang's model of learned heart rate change (1974, 1975). Lang has proposed that variables which have been shown to affect systematically learning motor skills should affect learning to increase heart rate but should not affect learning to decrease heart rate. Hypothesis 2(a), which predicted that the low sensitivity condition would show superior heart rate increases but equivalent heart rate decreases when compared to the high sensitivity group, was confirmed. While this hypothesis was confirmed, Lang's model still does not account for a number of previous findings. Five studies other than the present study found the predicted relationship among motor skills variables and learning to increase or decrease heart rate (Gatchel, 1974; Lang & Twentyman, 1976; Lang & Twentyman, 1974; Twentyman & Lang, 1980). Four studies reported finding motor skills variables which affected both learning to increase and decrease heart rate (Blanchard, et al, 1974b; McCanne & Sandman, 1975; Colgan, 1979; Williamson & Blanchard, (1979a). One study reported a motor skills variable which did not significantly affect learning to increase heart rate (Haynes, et al, 1977). Therefore, Lang's model has some explanatory power, but does not comprehensively account for the experimental findings which have been reported. It must

be concluded that Lang's model needs to be examined closely and revised to account for those findings which this model does not predict.

A possible explanation of the present study's finding of a difference between feedback sensitivity conditions in learning to accelerate heart rate has been raised (Williamson, et al, 1981). This interpretation of the data focuses on possible motivational changes produced by manipulating feedback sensitivity. This explanation proposes that the overall superiority of the low sensitivity condition's mean heart rate increase performance may be due to different levels of interest in the task. The low sensitivity group would have found the feedback needle more difficult to affect, possibly causing them to expend greater effort. The high sensitivity group may have found the feedback needle so easy to affect that the challenge of the task would have been considerably lower than that of the low sensitivity condition. If the high sensitivity group was less motivated, then perhaps this may explain the variability seen in their performance on the heart rate acceleration trials. Such motivational changes may have resulted in alternating periods of effort and non-effort, with resultant variability in heart rate acceleration performance. The absence of a difference between the heart rate decrease performances of the two sensitivity conditions might indicate that the sensitivity manipulation did not affect the subjects' motivation to the extent necessary to

separate the conditions' performances. This speculation as to the effect of motivational changes on heart rate decelerations draws upon the hypothesized role of situational and biological constraints in mitigating against the production of significant heart rate decelerations. The possibility that situational and/or biological constraints may affect the production of heart rate decelerations will be considered next.

An explanation of the differing results found for heart rate increases and decreases relates to biological and/or situational constraints on the production of large magnitude heart rate decreases (Williamson & Blanchard, 1979c). This explanation is based in part on Schwartz's observation (1974, 1977) that inherent biological constraints may limit the production of particular autonomic responses. Combined with suggestions (Cuthbert & Lang, 1976; Lang, 1980) that components of the biofeedback task, e.g. subjects working with a feedback display to produce a change in heart rate, may produce unconditioned heart rate increases, this alternative explanation proposes that biological and/or situational constraints counteract the production of large magnitude heart rate decreases. Williamson & Blanchard (1979c) accurately noted the extraordinary difficulty in independently determining the effects of biological or situational constraints. However, one study (Bell & Schwartz, 1975) has provided some indirect support for the role of situational constraints in producing heart rate

decelerations. This study had subjects record their heart rates at different times of day in the natural environment. These heart rate values were compared to laboratory heart rate values and it was found that laboratory heart rates during rest periods were close to the lowest values recorded in the natural environment, with the exception of just after awakening. The finding that resting laboratory heart rates are close to the typical low value of heart rates during an average day would cause one to expect production of large magnitude heart rate decelerations to be difficult. As these resting periods are typically used as baselines against which change is measured, baselines at the lower end of natural heart rate ranges would reduce the likelihood of further large decreases in heart rate.

The overall result of such situational or biological constraints would be a restricted range of heart rate values which could be produced. If this were the case, then a restricted range of heart rate slowing would reduce the likelihood of finding an effect of procedural variables on heart rate decreases. Only powerful variables would have a detectable effect within such a restricted range of values. Therefore, these powerful variables could significantly affect both heart rate increases and decreases. Less powerful variables would not be likely to produce significant heart rate decreases. This explanation is partially supported by the infrequent finding of mean heart rate decelerations greater than 5BPM (Williamson &

Blanchard, 1979b).

This explanatory hypothesis has some empirical support but requires further evaluation. However, this hypothesis raises intriguing questions about the role of biological and situational constraints in the development of heart rate control, particularly heart rate decreases. This hypothesis also presents a possible explanation for the failure to find a significant effect of feedback sensitivity on learning to decelerate heart rate in the present study.

Obviously, no conclusion about the above explanations of the present study can be drawn given the present data base. However, these proposed explanations do suggest directions for future research. The role of motivational factors in the production of learned heart rate changes appears to be one potentially fruitful area of research. Occasional studies have examined the role of incentives in heart rate change performance, but contradictory results and widely differing methodologies prevent any firm conclusions from being drawn (Williamson & Blanchard, 1979b). While the motivational state of subjects in biofeedback experiments is generally conceded to be an important component in maximizing the likelihood of the desired change, there is little agreement in the area of heart rate biofeedback as to the precise role of motivation and incentives. The role of motivation and incentives in learning to control heart rate is an area which needs to be investigated with an adherence to standard psychophysiological and experimental design

procedures so as to maximize the comparability of results from different laboratories. As was previously noted, the role of biological and situational constraints in learned heart rate change also appears to be a promising area of research. Such research affords an opportunity to explore the interrelationships among physiological systems which would be a crucial part of any thorough understanding of biofeedback.

The failure to find significant differences between the heart rate decrease performances of the sensitivity conditions of the present study is consistent with a number of published studies. The continued discovery that variables which significantly affect heart rate increases often do not affect heart rate decreases indicates that heart rate decreases are at least a quantitatively different response from heart rate increases. Lang's position that heart rate accelerations and decelerations are qualitatively different responses is not well supported by the literature but does point to a difference which needs to be explored. Both Brener and Lang's models of learned heart rate control are in need of revision concerning the learning of different responses. Increased understanding of the differences between learning to decrease heart rate and learning to increase heart rate would also aid any practical application of learned heart rate control by increasing the likelihood and/or efficiency of producing significant changes in the desired response.

It appears that the present study has found some of the limitations of feedback sensitivity as a feedback parameter. The finding that feedback sensitivity significantly affects learning to increase heart rate is reasonably well established, as the present study replicated the overall findings of Williamson, et al (1981). The absence of an effect on learning to decrease heart rate limits the value of feedback sensitivity as a subject of further heart rate control research. The absence of a demonstrated feedback sensitivity effect on self-control of heart rate further circumscribes the application of feedback sensitivity. The results concerning the effect of feedback sensitivity on learning to increase heart rate indicate that this variable should be considered when one is designing a feedback system with the goal of teaching heart rate increases. Also, feedback sensitivity should be reported as part of the procedural description of a study, in the same way that type of feedback (auditory, visual; binary, proportional) has become a standard element to report.

The discovery and refinement of the effect of feedback sensitivity on learned heart rate control indicates an important point concerning the general use of biofeedback. The initially unexpected effect of feedback sensitivity once again demonstrates that only an empirically grounded examination of biofeedback technique and technology offers a valid structure within which to use biofeedback. This fact mandates a continued fine-grained examination of both

methodology and theory in the area of learned heart rate control, as well as other areas of learned visceral control. In this way, we can continue to make progress toward the ultimate goal of greater understanding of the human ability to modify the functioning of the body.

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VITA

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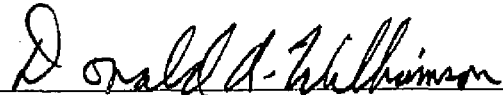
EXAMINATION AND THESIS REPORT


Candidate: John Edward Monguillot

Major Field: Psychology

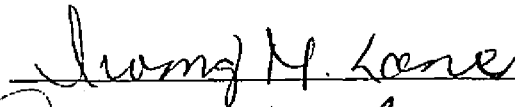
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Acceleration and Deceleration

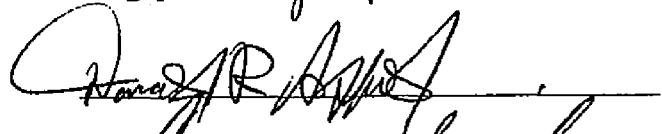
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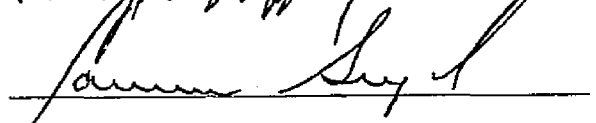

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